use the regulatory risk models by themselves to make assessments of whether disease occurred in an individual as a result of an exposure?

A No, the regulatory models which use the same general framework and data sources have a different focus. They also, for reasons I think I will try to explain later, have built in a number of assumptions that go beyond science that really get into policy, and they're really not focused on individuals. They are focused on populations only.

Q When you talk about the scientific risk models, do they or do they not have value in assessing whether people in the group have gotten sick as a result of exposure?

A Not only do they have value, they are essential.

Q Okay. Now, this doubling of risk, I want to come back to that for half a moment. If you do not have a doubling of risk -- let's say -- let's go back to our hypothetical and say that the increased risk was 1.5 or a 50 percent increase. If in its, you know, replicated consistent quality where you have a relative risk of less than 2, can you still make the statement that the disease in the group was more likely than not drug related?

A No, because less than half of the cases -- epidemiologists sometimes call this the attributable fraction or risk. But less than half of the cases for any risk -- any relative risk below 2 would've come from the drug. So, therefore, for any

individual it would've been less than a 50 percent chance that the disease came from the drug.

Q That's not my question. I want you to assume that the legal standard for causation -- I want you to make the assumption that the legal standard for causation, that is whether an individual got sick as a result of a drug, is a more likely than not standard. I want you to make that assumption. In your experience with risk assessment is there any way that science has to establish whether a given toxic material or a given drug more likely than not has caused disease? Is there any other way to do it other than to look for the doubling dose?

A Not to my knowledge. You rely on the epidemiology data, the dose response data, look at the doubling dose. I know of no other way to look at that question.

Q Okay. Has this been a subject that you specifically have been involved in personally in your work for the Federal Judicial Center?

A It's one of the topics the manual -- there's actually a scientific manual prepared to provide scientific understanding to judges. That's its purpose. It's really not a legal document, as far as I understand it. It's full of scientific chapters, and --

Q Is the testimony that you're giving regarding relative risk of 2 or more, is it consistent or inconsistent with what

that manual says?

A That manual --

MR. FINCH: Objection, Your Honor. This is invading the province of your court. He is offering testimony that I believe is intended to be later argued to demonstrate that the case law or the Federal Judicial Center manual requires what he is testifying to about risk assessment. That is a question of law, what is required to prove causation -- general causation, which is all this is about, in a case, and I think it's not proper for a expert witness to testify about a document that was created as a reference manual for judges.

THE COURT: Well --

MR. BERNICK: That's not the -- if I could explain, Your Honor. That's not the proffer at all. At some point there has to be an intersection between science and law, and all that I've done is to elicit that when it comes to their legal requirement, I've given that to him. This is what science has to say, and I think he very properly pointed to the Federal Center manual as being the effort of scientists to do precisely that. The manual doesn't dictate law, but the manual captures the view of scientists about how to meet the legal standards.

THE COURT: Well, actually, it was the effort of the Federal Judicial Center at the request of some judges for information about how to evaluate cases and -- that come before

them that deal with certain types of scientific evidence, and so it's neither. Nonetheless, I understand what the purpose of the manual is, and so I'm not sure where we're going with this. It's --

MR. BERNICK: I just want to establish that his testimony is totally consistent with the manual. That's all that I'm --

MR. FINCH: And that's part of my objection, Your Honor. His --

MR. BERNICK: Well, but there's no other person who can do that except for him.

THE COURT: I think that's a function of this Court, to determine whether his testimony and the manual are consistent, but what's the relevance? The manual is a reference guide. Whether the manual says A, B, or C is wholly irrelevant to the evidence that's going to be presented here. It's a guide for judges.

MR. BERNICK: That's fine. I don't want to belabor it, Your Honor. I think that the witness' testimony is people who put together -- I mean you're talking about consensus science. He can talk about consensus science, and the manual -- I think the witness' testimony is that whatever the original motivation was for the Center to commission it, they've solicited the input of scientists, and they --

THE COURT: They have indeed.

MR. BERNICK: -- it supports his credibility when he offers testimony that doubling dose is it. It supports his credibility, because there are a bunch of other scientists who apparently thought the same thing and helped put it in the manual. That's all the purpose of the proffer is.

THE COURT: I understand what is available in the manual. I understand the witness' testimony. I understand that there is a guide that the Federal Judicial Center has kindly put together to help judges who may not be informed about these matters or who may be in formed about these matters. The reference manual is a reference manual, and I -- and it has that validity or that value I suppose within the confines of the federal judiciary. I don't think this question really is relevant --

MR. BERNICK: That's fine.

THE COURT: -- to the area.

MR. BERNICK: That's fine.

BY MR. BERNICK:

Q I want to turn now to a different question which has to do with variations in dose, and I want to talk about how epidemiology deals not with a drug that's got a set dose but with a toxic agent or potentially toxic agent as to which there are variable doses or variable exposures. Are you with me?

A Yes.

Q And I want to kind of go through a similar hypothetical

exercise.

MR. BERNICK: And if we could show the Court GG-2011?

Q I want to take the Court in your testimony through on the strength of a series of assumed studies --

MR BERNICK: And at this point T.J., you can take this one down.

THE COURT: With respect to the evidentiary question that I reserved, Mr. Finch, at this point, based on the ruling that I've just made, I don't see any problem with the particular reference of the judicial manual on the chart that the witness has prepared. I think I understand that it is a reference manual and nothing else, and so to that -- and I will interpret the information on that chart -- the icon on the chart to be for that purpose.

MR. FINCH: That's fair.

MR. BERNICK: That was his only purpose.

THE COURT: All right.

THE WITNESS: May I clarify one point?

MR. BERNICK: At your peril of irritating the Court, but go ahead.

THE WITNESS: Well, it's just I think you might have said that I had something to do with the existing manual, and I did not.

MR. BERNICK: Well, I didn't mean to suggest that. I meant what you referred to.

THE WITNESS: Okay. I'm advising on the future, but I had nothing to do with the existing one. I think that may have leaked out but --

MR. BERNICK: Yea, it --

THE WITNESS: Okay.

MR. BERNICK: It could've.

THE WITNESS: Okay. I'm sorry.

BY MR. BERNICK:

Q On GG --

THE COURT: The judges appreciate all help on all scientific matters --

THE WITNESS: All right.

THE COURT: -- from anyone.

THE WITNESS: All right.

Q GG-2011. I've asked you when we put this together to assume the first study with a dose of 18, a risk of 3.5, and that it's statistically significant. That is the confidence interval does not include one. A second study that's got greater exposure, a greater point estimate for risk, also statistically significant; a third study, exposure that's much lower at 6, a risk point estimate of 1.4 not statistically significant; a fourth study that's got yet another dosage, also statistically significant positive, and then other studies with important limitations. And I've tried to display them on this chart, which is Exhibit --

MR. BERNICK: Does anybody know the exhibit number?

UNIDENTIFIED SPEAKER: Two 0 one one.

MR. BERNICK: Two -- no, not 2011.

THE WITNESS: Two 0 one one.

UNIDENTIFIED SPEAKER: Five eighty-one.

MR. BERNICK: Five eighty-one which is a magnetic board that we're going to be peeling things off.

Q So we have on the board the same hypothetical as GX-0581, and we tried to reflect the same results now with a series of plots. Are you with me on my hypothetical trying to do this promptly in order to get you out of here today and get us done with this?

A I'm completely with you, yes.

Q Okay. Now, I want to talk now about the question of dose response. In your experience in working with epidemiological studies does this kind of -- I don't want to say represent a typical, but is this an illustrative series of data points that would help you explain what epidemiology does with dose response?

A It is.

Q Okay. Now, I see that all these data points are plotted individually, and there's no line or no curve or anything about that. Is there such a thing now as scientific modeling of dose response?

A There is. One -- a model is simply -- usually a

mathematical not a graphical, but you can do it graphically, but better mathematically, a description of what you've observed, to try to take what you've observed and construct a mathematical picture, if you'd like, of what you've observed. So that's called modeling.

Q Okay.

A It goes on all the time in science.

Q Why did people in the field of risk assessment who aren't focused just on regulation or prevention but are focused on understanding the scientists -- science -- why do scientists do risk modeling?

A Because this relationship between exposure and disease is critical to understanding disease causation. It even has other uses, but that's critical to this causation analysis, yes.

Q It has been said in this court that once it's established that an agent can cause disease, sometimes what's referred to as generic causation, that that means that it's no longer important to see whether it can cause disease under other -- under various circumstances. I want you to assume that that's been said.

A Okay.

MR. FINCH: Objection. Lack of foundation.

MR. BERNICK: Well, no, actually, assertion was made here, but I'm asking him to make the assumption.

Q I want you to assume the position has been taken that once

an agent has been established to be causes of disease under certain circumstances, the only issue left is whether it caused disease on an individual basis. I want you to assume that that statement's been made. From a scientific point of view, is that correct?

A No, you've got to understand the dose response relationship and where exposures fall on that relationship

Q Just because --

A -- for individuals or groups of individuals, yes.

Q Let me put it to you again. Just because it's been established that an agent caused disease at high doses, does that mean it's been scientific -- it is scientifically established that it can cause disease at low doses?

A Absolutely not. No. That's why risk assessment came into being, because that mistake was being made a lot. We needed a framework, going back now 30 years to try to get better quantitative understanding of how the dose affected the rate of disease or the risk of disease.

Q I want you to assume for illustrative purposes that a model has been developed, and it finds a dose response relationship, the curve that is the one now indicated --

UNIDENTIFIED SPEAKER: Your Honor, may I stand up?

THE COURT: Yes, sir.

UNIDENTIFIED SPEAKER: I can't see --

MR. BERNICK: Oh, I'm sorry.

UNIDENTIFIED SPEAKER: -- from where I'm sitting.

Q I want you to assume that the relationship that's now been modeled as dose response relationship, as indicated on GX-0581, now that we've peeled off the strip, again for illustrative purposes does that assist you in explaining to the Court what modeling does?

A That's exactly what modeling does. As I said, it's usually expressed as a mathematical formula, but this graphic presentation is perfectly adequate. You're doing as best fit you can to the data trying to make that curve explain the data basically.

Q Okay. Now, we know that three of the studies -- Studies 1, 2, and 4 -- found positive statistically significant associations, and we see that the curve is drawn through them. And the title at the top is "Observations From Epidemiological Studies." I now want to go down to the last study that was there, Study 3, which finds at lower dose a relative -- a point estimate of 1.4 that is not statistically significant -- and so the record is clear, the blue model line ends at that data point, and that further, we have a couple studies, which are the other studies with important limitations, that fall towards the low end and are indicated also on the chart. And I'm -- with all that kind of out there, I want to ask you what it means to say that there is a zone of inference between the observed positive studies on the one hand and the negative

study where there was not a statistically significant association found on the other? What does it mean to say that that's a zone of inference?

A Well, we've got some measurements in this zone, but we've said they're not reliable, and then we have one at the low end which is not different from the background. So we've go uncertainty in this area about the true shape of the model. It becomes less certain in what you see at the higher exposures. So the statisticians who will be modeling there will admit that this is -- they're making some inferences that -- to fill in that model in that area. It's not -- it depends on each circumstance how certain that is, but it's less certain than the higher area.

Q When you're drawing the curve -- the dose response curve down below the last points of a positive association into an area where you don't have reliable studies showing a positive association, statistically significant, is it wrong -- is it scientifically wrong to draw that curve?

A No, you've got to be very careful in delineating them. Lay out all your assumptions for other people to look at, but it's done all the time, yes.

Q It's done all the time.

A Yes.

Q Is it proven scientifically that that relationship holds in that area? Recognizing it's not wrong to do it, I'm kind of

flipping the other way around. Under the hypothetical that I have given you is it proven that there is, in fact, a dose response relationship in that zone?

A It's not proven in anything like that same sense it's proven for the higher range of observation.

Q Now, I want to go even further into the unobserved range, which is the area that is below your last study, which is a negative study. That is you didn't find an association. It says, "unobserved range." What does that mean?

A Well, it is a range in which we either have not studied -- where we have some studied but have not found any significant finding. But usually it means once you're below that low point, it's a range that has not been studied.

Q Can science tell us in this area where it's below the last point of observation -- the last point of observation was negative. Can science tell us that there is a causal relationship of any kind? That is the agent being studied is causal, below that last study in that unobserved range?

A Can science tell us? No.

Q Can science tell us that?

A No, and science remember is basically empirical. You have to have data and a model to describe the data, but once you get beyond the data you are not in the realm of science. You're in the realm of hypothesis.

Q Does the fact that there's not observation in that area --

does that rule out the possibility that there is a risk?
A It rules out a significant risk, a large risk, certainly, but it's possible that there may be some risk as you go below this region. We don't know where it ends. We don't just have knowledge there, but you -- we wouldn't assume that the risk just ends at the point where you happen to measure. But it's certainly going to be small, and as I say, we really don't know how it behaves, what the dose response looks like.
Q Under the hypothetical that I've given you, which is at the high or high dose end of that unobserved range, there is an observation, and it's a negative one. That is there's no an association. What does -- under my hypothetical what does the available scientific evidence say about whether there is real risk in this unobserved range?
A If you interpreted this scientifically, you would say there is no observation of excess risk at 1 point -- at the dose -- the lowest dose that we have studied here, and it could -- that no effects dose actually could be even higher as you go up that curve. We're not quite sure where the dividing line is, but that certainly is a no -- we call it in toxicology or epidemiology a no observed adverse effect dose.
Q Now, I want to talk about regulators, and I want to talk about regulators, because we said back at the outset, remember, you made a distinction between risk assessment models that are used by regulators and risk assessment models that are purely

scientific. You made a statement they're not necessarily the same.

A Right.

Q When you deal in the real world of talking to regulators, will regulators necessarily consider this kind of data point that is a negative study at an area of positive dose? Will they necessary consider that to be definitive?

A Not necessarily. They might. If it were very well established through many studies, they would find that definitive. They tend to err on the side of caution, and in most cases they would say, well, there could be risk below this area. We have a public health protection mandate that makes us want to look at where populations are exposed typically at very low doses.

Q So they might say --

A So they make the assumption that, well, there might be something going on here.

Q They might say there is not a point at which science can be considered sufficient to say no risk.

A Correct.

Q Okay, and have you written about that yourself?

A Quite a bit, yes.

Q Okay. Let's now talk about what the regulators do.

MR. BERNICK: And I'm going to move through this quickly, Your Honor, in an effort to finish here. I want to

show the Court Exhibit GG-2023.

Q Could you use Exhibit -- would GG-2023, Dr. Rodricks, assist you in explaining what regulators often do in this unobserved range area?

A I think it does. Yes, I hope it does.

Q Okay. Could you tell the Court what that demonstrative shows?

A Well, regulators are concerned about very low risks, risks that are well below the range of observation. They might be concerned about relative risks of let's say not 1.4 but 1.0004. They interpret their legal mandates to be highly protective to want to protect populations at very, very low levels of risk. So they can't measure these. These are below the range of observation, so it's become practice to assume -- to apply so called extrapolation models. Extrapolation means you're going from the data into the unknown. And we don't know the truth about how the dose response curve varies below that zone of observation. We just don't know.

So regulators look at the possible ways it might vary and have selected routinely what I tried to show on this here with the yellow line, where there's a straight line drawn from the lowest observed -- or the lowest unobserved risk down to zero risk. And that line is chosen, because we have quite convincing argument at least based on statistics that any real risk that's down there in that zone would be -- it would be

very unlikely to be greater than that risk. You can't prove that absolutely, but with very high confidence we could say that's going to put an upper limit on the risk. It's not going to be greater than that. It could be a lot less, and I tried to show with one of the lines that it could actually be zero. It could drop rather quickly to zero risk. We just don't know.

Q Have agencies like the EPA been candid in expressing the kinds of limitations that you have now articulated to the Court?

A I think they have. I think the -- if you look at what agencies say, they're pretty honest about what they're doing here. They do it for public health protection purposes. I do it every day for the same purpose but not for disease causation. These are very, very small risks, and they are known risks.

Q Are you familiar with the Risk Assessment Guidelines of 1986 as issued in August, 1987 by the EPA?

A Yes.

Q Is that a government study -- government-issued document by the EPA?

A The EPA describes how they do risk assessment and their assumptions, yes.

Q Is that a formal pronouncement of an authorized agency of the United States?

A It is.

MR. BERNICK: We offer GX-580, which is the Risk Assessment Guidelines of 1986 issued in August of 1987 by the US EPA.

THE COURT: Do you have it marked as an exhibit?

MR. BERNICK: It's Exhibit -- yes, it is. It's Exhibit GX-580.

THE COURT: Any objection?

(No verbal response)

THE COURT: It's admitted.

UNIDENTIFIED SPEAKER: No objection from the Future Claimants.

MR. BERNICK: I'm sorry?

UNIDENTIFIED SPEAKER: No objection.

MR. BERNICK: Okay.

THE COURT: It's admitted.

BY MR. BERNICK:

Q I'm showing you GG-2024. Is this a quote taken from the EPA's pronouncement?

A Yes, it's a direct quote.

Q It says, "It should be emphasized that the linearized, multi-stage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanism of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk. The true

value of the risk is unknown and may be as low as zero." Do you see that?

A I do.

Q Is that consistent or inconsistent with your own testimony about what regulators often do in risk assessment framework?

A It's completely consistent. The -- this technical term, linearized multi-stage procedure, is just another name for that straight line that I described.

Q Let's now -- I want to take you through the last series of questions about these data points, and then I want to close with a few questions and turn you over to their tender mercies at the other side of the courtroom. If we go on the dose response axis -- on the dose axis, under our hypothetical we go from zero exposure all the way to the exposure associated with that negative study. Based upon science -- scientific data you say that that dose actually carries with it any risk, based on science?

A The dose corresponding to the risk of 1.4?

Q The dose -- yes, as --

A I'm sorry.

Q All doses that lead up to that data point of 1.4. You say that any risk has been demonstrated be associated with that dose?

A There's no demonstration of risk here. That's -- there is none.

Q If we now go -- and we're on our curve -- to doses that are greater -- doses that go all the way up to your positive results, that curve looks nice and neat and solid, like there's every little bit -- every -- like we went in with a microscope -- every little bit more would carry with it some additional risk. Is it true, necessarily, that because you have a dose response model, that every item of increment up that curve, including in the observed areas, carries with it an increment of risk?

A No, you have to be very careful as you go up the curve to see where you are. You have to take into account if you -- like with the mathematician or bio-statistician would call the imprecision of that curve. Remember, it's built from a few points with some inferences, and there's a confidence interval actually on the whole curve that tells you something about what I call the precision of that analysis. It's not a real precise curve, so it depends on the increment of dose whether or not it becomes a statistically significant increment.

Q Okay. I'd like to show you GG-2017. So, I want to ask you a new hypothetical here. So, we assume that there's an exposure from some other source that puts a group along the dose response curve as indicated in 2017 at a dose of 17, and I want you to assume that at that dose there's data that says that, yes, there is a positive statistically significant risk. That's the beginning of the hypothetical, is we have a dose

from another source that carries with it real risk.

I now want to show you 2018 and in 2018 we've now added more, we've gone from 17 to 22. Can you tell from the way that that chart is drawn, where we have another study 22, can you tell whether or not that incremental dose has been scientifically shown to present an observable increased risk?

A I can tell, yes. We're looking now -- we started with a does of what we call what, 17 and now we're asking about the increment up to 22 for this curve.

Q Yes.

A Is that a significant increase, you could --

Q Is that an observed significant increase?

A Oh, I'm sorry, it's not observed.

Q Okay. And why do you say it's not observed?

A Well, we don't have an observation point at that point. We have a model and the model would say, we could calculate an increase but you also have too look at the confidence interval on that model.

Q Okay. Given the model can you say it is the model tells you that, in fact, there is an increase?

A Not in fact.

Q And tell the Court now, making reference to that chart, why that is so.

A The values predicted by the model with confidence intervals, do not differ statistically. So, that's a

reflection of how precise this curve is, if you like, so you could say, yes, you could calculate that one risk is X and one risk is somewhat above X but statistically they are indistinguishable.

Q So, if we go over to the chart over here, what you're saying is that the model itself has confidence intervals. That within 95 percent confidence interval, the truth is somewhere in between, but it may not be at exactly these points?

A That's one way to put it, yes.

Q And, if you go from the point at 17 to the point at 22, what's the key feature of the confidence intervals that tells you that that is not statistically significant?

A Well, they overlap. You remember the discussion earlier of when it overlapped, the value of one or not, same idea here. Do they overlap? If they overlap then they're really not distinguishable.

Q Okay. Now, what if you wanted to, I want to ask you, let's go to 2019. Let's look at 2019 and we've shifted further up from 17 to 26. Tell me now whether the model with its confidence intervals tells you that there's been an observed increased risk?

A In this case, with this example, you are clearly in a different zone and the confidence intervals do not overlap, so you'd say here, that difference, that increase in dose gave rise to a statistically significant increase in risk.

Q Okay.
A So, in this case you've reached that point. You've sort of overcome the imprecision in the model, the difference are large enough that the imprecision doesn't matter that much.
Q Okay. We now want to go to the question, have you become familiar, have you had exposure in your work to the term substantial, that is a substantial contributing factor?
A I have, yes, seen that term.
MR. FINCH: Objection, Your Honor, to the extent he's offering a legal opinion, or legal testimony of what it means to be a substantial contributing factor. I think it invades the province of a court.
THE COURT: So far the only question is, has he heard the term and he said.
Q Okay. From a scientists point of view, do you have a way of looking at does response relationships, do you as a scientist have a meaning for the term substantial when it comes to incremental risk associated with incremental dose?
A Well, I use the term, for the risk doubling concept, that to me, any risk doubling, would be a substantial increase in risk.
Q And, why do you use risk doubling as a benchmark for substantial?
A For the same reason I described earlier, that once you've reached that point, you can say that most of the cases you

observed in that population are due to the exposure not the background.

Q Last few questions. Tell us whether or not you find that risk assessment is as reliable method based upon your expertise for determining causation by an agent for disease in groups of people. Is it a reliable method?

A It is reliable and as far as I know, it's the only method for doing that.

Q Let's ask you the same question about dose. Tell us what your view is as to whether risk assessment is a reliable scientific method for determining the dose at which an agent is proven to cause disease in groups or individuals.

A I believe it is, yes.

Q Are you aware of any other method that reliably enables a determination about whether a given toxic agent causes disease in groups of people and the dose at which it does?

A I am aware of no other method.

Q Let's talk a little bit about acceptance. Tell us whether or not risk acceptance is accepted within the scientific community for the purposes I've just asked about, that is determining causation and dose of causation for groups of people and individuals.

MR. FINCH: Objection, lack of foundation. He's asking about the -- for determining causation in groups of people, he's asking about it in the context of a tort case and

I don't think this witness is entitled to answer that.

MR. BERNICK: Your Honor, I asked him no such question. I asked him as a scientist within his community of sciences. I mean, scientific causation. That's my question. As a scientist tell us whether or not risk assessment as you've described it, is accepted within your scientific community as being a reliable method for determining causation in groups and individuals.

THE COURT: I think he's competent to answer that question and to offer an expert opinion about that. The objection is overruled.

Q Yes, go ahead.

A The answer is yes, for sure. I would add one small modification. Some scientists who go through the process and may not label that process risk assessment, but the steps of the analysis that I showed in risk assessment they do follow. So, I would say with that very minor twist, yes, the answer to the question is definitely yes.

Q Are you aware of any other scientific method that is accepted as reliable for those purposes?

A I'm aware of no other method.

Q Let's talk about consensus. Does the term scientific consensus have meaning to you within your field of risk assessment?

A Yes.

Q Okay. And what does scientific consensus mean?
A It means that, technically, it means that every scientist accepts the method. That's really the case with any scientific principle, but it means a vast majority of scientists who work in the area will accept the method, I think.
Q Okay. Is this, again, if I ask the question, the same question, that is the use of risk assessment reliable for the purposes of determining causation of a toxic agent, is there a formal consensus that's been announced to that?
A A formal one?
Q Yes.
A No, no, not that I know of.
Q Okay. Do you believe as an expert in the area that there is essentially, not complete consensus, but substantial consensus, in fact?
A I do. I do.

MR. BERNICK: Okay. I have no further questions.

THE COURT: Mr. Finch?

MR. FINCH: Can we take a short recess to reset this courtroom?

THE COURT: Yes. Would you tell me just how long you two think you will be on cross-exam?

MR. FINCH: I'm probably an hour.

MR. RASMUSSEN: And I'm probably half an hour.

THE COURT: I do not think we will finish tonight.

MR. BERNICK: I just really -- (indiscernible), Court's decision, we really committed to Mr. Rodricks that we would get him out today, if at all possible.

THE COURT: I cannot keep staff her until seven o'clock tonight. I just can't do it. And that's how long it will take.

MR. BERNICK: It would be six, Your Honor.

MR. FINCH: It would be six, Your Honor.

THE COURT: Well, we'll stay until six, but you folks have to limit your examination and Mr. Bernick, that includes you on redirect.

MR. FINCH: Now wait a minute, wait a minute, Your Honor.

THE COURT: Until six. If we're going to be done at six, we are going to leave at six.

MR. FINCH: That's fine, but if there is additional topics that Mr. Rasmussen has --

THE COURT: For us to leave at six, you have to limit your examination, so that we can be finished at six, otherwise you need to tell the witness so that he can make other arrangements, so that he can be here again, another day. That's what I'm saying.

MR. BERNICK: I mean, this is an ongoing problem that Your Honor (indiscernible) but we did get specific time estimates. The time estimate was given to us in advance for

cross-examination was between an hour and an hour and a half for both cross-examinations.

THE COURT: That's what you just heard.

MR. FINCH: And that's what we just said, but we --

MR. BERNICK: Well, there's always -- turns out to be, just like they did with the cross of the last witness, it's more than that and I'm obviously concerned, we go an hour and a half and then it'll all be used up and we still have to bring him back.

THE COURT: Well, gentlemen if we continue to bicker about how long it's going to take, you're going to cut into the time that you have available. In order to be finished tonight, we are going to leave at six. So, plan yourselves accordingly. I hope, sir, that we will be able to excuse you.

THE WITNESS: Thank you.

THE COURT: Okay. We're in recess for five minutes, and literally five minutes.

(Short break in proceedings)

COURT CLERK: Judge wants to come on.

THE COURT: Please be seated.

COURT CLERK: Please be seated.

THE COURT: All right, Mr. Finch, go ahead.

MR. FINCH: Nathan Finch for the Asbestos Claimants Committee.

CROSS-EXAMINATION

BY MR. FINCH:

Q Good afternoon, Dr. Rodricks.

A Hello.

Q Could you turn on the ELMO please. This is one of the slides that Mr. Bernick showed you. Is it correct that to do this kind of an analysis you need an accurate measure, accurate measure of the dose received?

A Generally, yes. Yes.

Q You need a quantitative measure of the dose received?

A Well, if you're asking, you want to know what the risk might be at a given dose, you --

Q Yes.

A -- yeah, you should have a good idea of what that dose is, yes.

Q And, you also need an accurate identification of what the background rate of disease is, or what the rate of disease could be caused by their factors to make a risk assessment?

A Well, in construction of this dose response created, you've already taken that background into account. So, the dose response take the background into account already, and now we're just asking the question for a given dose what calculated risk would you find, what elevation.

MR. FINCH: I don't know if Your Honor can hear but it might be good if -- can you hear okay?

THE COURT: I can -- are you having trouble hearing?

MR. FINCH: It's hard to hear back here.

THE COURT: All right.

Q All right. Here you have two different measures of dose --

(Microphones malfunctioning)

THE COURT: I'm sorry there have to be two microphones on somewhere. (Indiscernible).

(Pause)

THE COURT: I'm sorry, Mr. Finch, go ahead.

Q In this hypothetical you have two different observed measurements of dose with 95 percent confidence intervals that don't overlap, correct?

A Correct.

Q Okay. And, you would say that that is a significant increase in risk if the confidence intervals don't overlap, correct?

A Yes, that is a measurable increase, significant increase in risk, at that point.

Q You've mentioned the concept of relative risk. There's also a concept in epidemiology, a risk assessment called Odds ratio, correct?

A For a particular kind of study, yes, that's correct.

Q And, would you agree that if the disease is relatively rare in the general population, about 5 percent or less, the Odds ratio is a good approximation of the relative risk, which

means there -- excuse me, that the Odds ratio is a good approximation of the relative risk?

A Yeah. The Odd ratio is used in something called case control studies which are generally focusing on rare conditions and they're calculated in a somewhat different way than relative risk but, again, as you said for low risk they are considered to be -- they are approximately the same measure, yes.

Q As relative risk.

A Yes.

Q Okay. To save time, I was going to draw like Mr. Bernick, but I'll use the ELMO. He likes to draw. All right. I want you to assume from my hypothetical that there's a carcinogen where you have data points that shows at a dose of less than 0.5 units of measurement, the relative risk is 1.1 and the 95 percent confidence interval is 0.8 to 1.7. At a dose which is greater than 0.5, but less than 1, the relative risk is 4 and the 95 percent confidence interval is 1.7 to 9.7.

THE COURT: I'm sorry, I can't see the left column. What is the second line, please?

MR. FINCH: The second line is 1.0 greater than dose, greater than 0.5. Do you see that, Dr. Rodricks?

THE WITNESS: I think I understand what you -- yes.

Q Okay. And the 95 percent confidence interval is 1.7 to 9.7.

A Right.
Q And the third line you have does less than 10, greater than 1 and the relative risk for that is 4.0 and the 95 percent confidence interval is 2.2 to 7.2. Do you see that?
A I see it -- could you move the paper just a little bit?
Q Which direction?
A Well, because you're cutting off the confidence interval there. That's --
Q There.
A Okay. I think I see it.
Q Okay.
A Would you agree with me if that's what the data shows, that would be a significant increase in risk if you went from does 0.5 to a dose greater than 1?
A So, you're starting with a dose at .5, is that the premise?
Q You're starting with a dose -- of a dose at less than .5, the relative risk is 1.1.
A Right.
Q When you move the does greater than .5, but less than 1 the relative risk is 4.0 but you have a 95 percent confidence interval of 1.7 to 9.7.
A So, that would be a statistically significant increment in the risk, yes.
Q So, going from 0.5 to between -- less than 0.5 to between

0.5 and 1 would be a statistically significant increase in the risk.

A In the datas that you've written here, yes.

Q And, going from a dose of less than 0.5 to above 1, would be substantially significant increase in the risk, correct? It would put all of the confidence intervals above the doubling dose of 2.2, correct?

A Well, you're almost correct.

Q Okay.

A Relative to the original .5, the increase going from 1 to 10 that range, you have a significant increase above the .5, but not above the .5 to 1 range. They're in the same range. So, those are indistinguishable.

Q Okay. But the risk is certainly greater if you go from -- once you get above the 0.5 threshold.

A Yes.

Q Okay. And would you agree that as a matter of statistics it is possible, is it not, that two measurements which differ, which have overlapping confidence intervals are still -- can be significant, correct?

MR. BERNICK: Objection to the form of the question. as too ambiguous.

COURT CLERK: Come to the mike.

THE COURT: Sustained.

MR. BERNICK: Objection to the form of the question.

He just set out a very detailed hypothetical and now, I don't know if that has a reference to the hypothetical or something else.

MR. FINCH: No, I'm just asking as a matter of statistics, the -- even if there is an overlapping confidence interval at the 95 percent confidence interval, you can have one dose that's higher than another. And it may be significant, it just may not fall within the statistically significant.

MR. BERNICK: I would object to the form of that question. I don't understand how can it be significant but not statistically significant, and it's ambiguous.

THE COURT: I apologize, but I don't understand the question either. Could you please rephrase it?

MR. FINCH: All right. I'll back up.

Q There are -- this is a higher dose than this.

THE COURT: That's not on (indiscernible), Mr. Finch.

Q This is a higher dose than this, correct?

A Yes, that's correct.

THE COURT: Wait.

THE WITNESS: I'm sorry.

THE COURT: For the record, you're going to have to say what you're pointing at.

Q I'm pointing to the chart that's been marked GX 05, GX 0581. And, it shows a data point of 4.9 and 3.2 with a range

of 3.9 to 5.8 versus 2.2 to 4.0. That's what we're looking at.
A That was my example, yes.
Q Okay. And the 4.9 dose is higher than the 3.2 dose, correct?
A Well, just the way you said it, those are not doses. The doses that give rise to those risks --
Q The doses that you -- excuse me, the doses that give rise to that risk, the 4.9 risk is higher than the 3.2 risk, correct?
A Yes. That's he measured value from the study.
Q And, each time you add to the dose, the sign of the risk, meaning whether it's positive or negative, increases, correct?
MR. BERNICK: Objection to the form of the question. Are we talking about the point estimate, are we talking above overall risk?
MR. FINCH: I'm talking about the point estimate.
THE WITNESS: So, you're asking, let's say we begin at the dose that gives rise to the 3.2 on my chart?
Q Yes.
A And, now we're going to add to that?
Q Yes.
A What I tried to say is that you could calculate from the blue line in the model that there is some increment but you also have to consider the imprecision in that blue lien to see whether that increment is statistically. So, the increment all

the way -- in this particular example, all the way up to the next measured point, none of those increments would be statistically significant relative to the 3.2. That was what I tried to say.

Q Okay. But the additional increased in the dose are additive to the risk for a dose response disease, correct?

MR. BERNICK: Objection to the form of the question.

COURT CLERK: Speak into the microphone.

MR. BERNICK: Objection to the form of the question. I don't know how that's different from the prior ones he just answered. What's the difference?

MR. FINCH: The difference is that he was asking about point estimates, now I'm asking about the overall curve.

MR. BERNICK: Then what's -- Your Honor, I don't mean to be obstructive, I don't know what the precise question being put -- the overall curve what?

MR. FINCH: The overall dose response curve.

THE COURT: Would you restate the question, Mr. Finch, please.

MR. FINCH: Sure.

Q Would you agree with me that for -- strike that. I'll rephrase the question when I get to the document. May I approach?

THE COURT: Yes. Thank you.

Q And, Dr. Rodricks, this is an article which you wrote in

2006, correct?
A It is, yes.
Q Could you turn to Page 40.
A Okay. Yes.
Q Would you agree with the statement that, and this is in the first paragraph, about seven lines down, "Exposure analysis which is a critical component of the valuation of causation may be relatively straightforward in the case of certain products but may become exceedingly complex if it involves reconstructing exposures arising from contaminated environments, especially where there is evidence that the exposures have been ongoing for long periods of time. Experts in exposure violation come from many disciplines, chemistry, chemical, environmental and engineering modeling of the movement of chemicals through the air and water into the food supply and industrial hygiene, and even a partial discussion of the nature of their work would significantly distract from the principle concerns of this paper. For this presentation, it is assumed that accurate medical diagnosis can be achieved and that reasonably accurate estimate of plaintiffs exposures can be derived." Do you agree with that?
A You're asking me to read what I wrote here?
Q Yes.
A Yes, I do. I mean, what I said is, I'm not going to discuss exposure assessment in any detail. I was going to

focus on what I earlier described as the causation analysis, the dose response analysis, but I'm just saying that you cannot complete an assessment without some measure of the exposures as well.

MR. BERNICK: Your Honor, I'm happy to have this --

COURT CLERK: Speak into the mike please.

MR. BERNICK: Sorry. I'm happy to have the examination continue with this document, but there was strenuous objection to our getting into any feature of his interface with the tort system or tort lawsuits. This article is in the legal journal and it's all about it.

THE COURT: Then on redirect you'll have a ball.

Q On Page 42 you write, you're scribing in the article, "Part 2 addresses how public health and regulatory scientists evaluate the potentially adverse health consequences of chemical exposures within a framework called risk assessment. That same framework is useful for evaluating disease causation in individuals but we shall see that some of the types of scientific evidence used commonly in the regulatory context may not be appropriate for evaluating causation in individuals." Do you agree with that?

A Yes.

Q In Part 1, Page 42 you write that there is a -- this common understanding has resulted from half a century of scientific dialogue --

A I'm sorry, could you tell me where we are?
Q Yes. Under the --
A Oh, yes, I see. Go ahead.
Q Much of it guided by many expert reports on this topic issued by various arms of the national academy since the early 1980's. What you're talking about is scientific -- scientists undertaking toxicological risk assessments in a regulatory setting, correct?
A In that case, yes, that's correct.
Q Okay. And that there's a common understanding among scientists, although they may disagree about the particulars of the way to conduct toxicological risk assessments in a regulatory setting, right?
A Correct.
Q Okay, but then you write, "No such history of scientific discourse has informed the risk assessment process as it relates to disease causation in individuals and it is difficult to discern anything remotely like a scientific consensus on how different types of scientific evidence should be used in assessments". I take it you believe that to be true?
A Yes.
Q Then you state, what is presented here, and here this is the paper you're talking about evaluating disease causation in humans exposed to toxic substances and a framework for doing that from a risk assessment perspective, right? That's what

the paper is about.
A Yes, it is, yes.
Q Okay. And when you say what is presented here might represent the thinking of most scientists, but no scientists writing on this topic would claim that it represents a consensus or that there are no alternative approaches that might have utility. I take it you believe that to be true.
A Yes. Let me explain. That has to do with the use of different kinds of evidence within the risk assessment framework. I did say, and I think I testified, that the framework itself, as a method, I do believe there is consensus on. I was talking about a slightly different topic here.
Q On Page 45, you write, "Toxic responses are a function of the magnitude of the dose and it is established with high certainty that toxic responses do not appear until a so called threshold dose is exceeded. They increase in incident severity, or both, as the dose increases above the threshold. But we are protected from harm if the threshold dose is not exceeded." Do you agree with that?
A Yes.
Q Okay. Then you write: "This threshold hypothesis well documented although it is not possible to claim the hypothesis holds for every chemical or type of toxicity. In fact, as shall be seen below, there is evidence it may be incorrect for certain types of carcinogens." Do you agree with that?

A Yes, that's correct.
Q Now, there is something called the World Health Organization International Agency for Research on Cancer?
A Yes.
Q It's called IARC?
A Correct.
Q The IARC lists 95 substances, mixtures or occupations as causally related to cancer, based on epidemiological data?
A That sounds right, yes. The latest listing.
Q There are different categories of carcinogens, correct, or classes?
A Well, the categories of evidence. Is that what you're referring to?
Q No. The IARC divides, the I-A-R-C divides carcinogens into various categories, correct?
A Well, I just have to be sure what you're talking about. There are different classes of chemicals but there are also different classes of evidence. So, you have to tell me which of those you're referring to.
Q The chemicals.
A The chemicals. Okay, yeah, they do.
Q Okay. Turn to Page 49 of your article.
A Yes, I have it.
Q Do you agree that the IARC, I-A-R-C list of carcinogens is promulgated by one of the most authoritative scientific bodies

that evaluate carcinogenity?

A Yes. It's in that category.

Q Asbestos is a category 1 carcinogen, correct?

MR. BERNICK: Objection, goes beyond the scope.

MR. FINCH: Your Honor, this is an article he wrote that I can cross-examine him --

MR. BERNICK: It makes no difference whether it's a article he wrote, it's on a subject --

COURT CLERK: Speak into the mike.

MR. BERNICK: -- if it's on a subject that goes beyond the scope of my direct examination, if they want to call him adversely in their case, and try to make an expert on asbestos they're more than welcome to do that, there may be an issue about that but that's what they're doing now, is to examine him on a subject that was not the subject of direct examination.

THE COURT: This is outside the scope of the direct and so far, unless you're going to raise some credibility issue, which I'm not sure what that is at the moment, based on where you've gone so far, I think it is outside the scope.

MR. FINCH: Okay.

Q Would you agree with me that categories -- materials that have been listed as category 1 or category 2 by IARC, one can presume that general causation is established?

MR. BERNICK: Objection. Objection to the form of

the question of the word presume, but, again, this is just a Trojan horse for exactly the same question, because he knows that he's going to pick up in that the categorization of IARC for asbestos. So, what he's really asking this witness to do is to vouch for the accuracy, I'm not sure how that came out, is to vouch for the accuracy of IARC's categorization and his characterization of asbestos. I'm not --

MR. FINCH: That -- I'm sorry. That was not my --

MR. BERNICK: That goes beyond -- well, I don't know what else the proffer is.

MR. FINCH: That was not my question. My question -- this was, all of your testimony about risk assessment relates to general causation in the first instance and into specific causation, correct?

THE COURT: And, in fact, you objected to his being proffered as an expert when it came specifically to asbestos, and now you're opening the door, Mr. Finch.

MR. FINCH: I'm not asking him about asbestos.

THE COURT: You are asking him about asbestos. The first question was, is asbestos a category 1 carcinogen and the second question was, if I look at category 1 carcinogens, is there a presumption that certain things will happen. You are definitely asking him about asbestos and you are outside the scope of your own objection to this witness being able to testify as to an expert opinion on that very subject.

Q Leaving asbestos completely out of it, would you agree with me that from a risk assessment perspective, if the International Agency for the Research on Cancer has labeled something as a category 1 carcinogen, that that is sufficient to establish general causation?

A I would agree with that.

MR. BERNICK: Objection to the form. Objection to the form of the question, including the use of general -- it really is the same thing all over again, Your Honor. It's just kind of a game here about what we're doing

MR. FINCH: Your Honor, it's not the same thing all over again. Your Honor, I said take asbestos completely out of it.

MR. BERNICK: Well, then what's the purpose for the proffer?

THE COURT: Exactly.

MR. FINCH: What's the purpose of the witness who is not testifying -- he was talking about general causation and specific causation, Your Honor. I am getting -- this is a fundamental question going to the question of, if something is -- if this witness believes that something listed on the IARC registry as a class 1 carcinogen, whether from a risk assessment perspective general causation has been proven.

THE COURT: All right. That's within the field of his expertise. Overruled.

MR. BERNICK: Well, okay, Your Honor. I really think that if he wants to try to tie this witness' testimony to IARC, he would have to present the foundation that says that IARC's classification bears upon the criteria that he used and they haven't done that yet.

THE COURT: He has not tied anything about IARC to this case. In fact, there has been an objection to this witness saying anything about asbestos in the case. The witness was called to explain the risk assessment models which he has done. But, nonetheless, this question appears to be within the scope of his expertise. The objection is overruled, would you state the question again so the witness can answer it, please.

Q Would you agree with me that if IARC, the International Agency for Research on Cancer has classified something as a category 1 carcinogen, in your opinion as a risk assessment expert, that is sufficient to establish that the general causation of that carcinogen?

A Just to explain a little bit, generally, yes, yes. Substances or other things get into category 1 when the IARC committees look at the evidence, the way I described, they look at the quality of the studies, they apply the Hill criteria and make a judgment, then, based on the criteria about whether the associations are causal.

I think it's generally reliable, but I think I would -- if

you ask me about any specific agent, I would wan to look at it separately to see if they've done it properly. If I wanted to make a serious inquiry. But generally, I would assume that they've done ir properly, but, again, serious inquiry on any specific agent I'd want to look at the underlying data myself.

Q Okay. Could you turn to Page 57 in your article?

A The first paragraph you write, "It is not possible to determine, except in unusual circumstances, which actual and individuals in a population are at the high end of sensitivity and also at the high end of exposure." Do you agree with that?

A Again, we're talking about a population, that is correct.

Q Okay. All right. Then on Page 58, under heading 3 you talk about general and specific causation in actual individuals.

THE COURT: I'm sorry, what page, please?

MR. FINCH: Page 58, of the same article. Actually, before we get there, on Page 57 you write, "a final point regarding the regulatory risk assessments is that the so called safe levels are derived from the epidemiological or experimental toxicity data by the use of assumptions that have the effect of placing those levels at a very small fraction of the observed threshold levels, do you see that?

A I'm sorry, I guess I don't. Where are you? On --

Q I am --

A Start with 57, sorry.

Q 57, I'm sorry.
A And, again, could you just cite the final point?
Q Yes. You're writing a final point regarding the regulatory risk assessments is that so called safe levels are derived from the epidemiological or experimental -- you see where I'm reading from.
A Yes.
Q Okay. That was the subject of some of your direct testimony today, correct?
MR. BERNICK: Again, I object. You -- you read half a sentence that ends in a note. I mean, is your intention to ask just about the first part sentence, or the whole sentence?
MR. FINCH: I will ask about the whole sentence.
Q Mr. Bernick pointed out that it ends in footnote, correct?
MR. BERNICK: Well, actually, even the end of the sentence, you continue on. By the use of assumptions that have the effect of placing those levels, at a very small fraction of the observed threshold levels, and then there is a footnote.
Q Okay. I take it you agree with the text, that's my question Dr. Rodricks.
A Yes.
Q All right. And in the footnote you write "Regulators assume in the absence of evidence to the contrary, that carcinogenic chemicals (indiscernible) mechanisms that disobey the general threshold rule for toxicity, this does not

translate to the sometimes expressed view that any level of a carcinogenic exposure can cause cancer. Citing sources. Instead, it means only that any exposures to carcinogens will increase cancer risk and that the magnitude of the risk increases with increasing exposures". Did I read that accurately?

A Yes. And that's what I described earlier. That straight line, if you recall the straight line extrapolation, the one that was yellow, that assumes no threshold. That assumes that even the smallest dose will increase risk, that's an assumption that regulators make for carcinogens, unless they have some evidence that it's wrong.

My point here was that that no threshold idea is commonly interpreted to mean any exposure that causes disease. And that is completely fallacious.

Q Okay.

A That's not what the no threshold idea means, at all. So, that's the point I was trying to make here.

Q Okay. All it means is that any exposures to carcinogens will increase cancer risk and that the magnitude of the risk increases with increasing exposures.

A Right. The assumption that -- the threshold assumption leads to that picture, yes.

Q Okay. Now, on Pages 58 and 59, you have a framework for evaluating general and specific causation. Do you see that,

Dr. Rodricks?
A Yes.
Q Okay. And you break it out into a series of Romanettes. The first is, to what chemical was the plaintiff exposed, then the second is, is there sufficient evidence in the scientific literature to support a causal relationship between exposure to the chemical and the specific type of injury or disease the plaintiff has incurred. That's general causation, correct?
A It's usually called that, yes.
Q Okay. And so, if the answer to number 2 is no, that usually ends the inquiry. If the answer to number 2 is yes, then the inquiry continues and that's where you get into the area of specific causation, correct?
A Yes.
Q Okay. Then Romanette 3 is, what is the likelihood that someone having the plaintiff's characteristics, age, sex, race, smoking habits, alcohol consumption rates, et cetera, would have the specific injury or disease, in the absence of exposure to the suspect chemical. That's what is known as specific causation, correct?
A Well, it's one of the issues, but this is the dose response relationship and what the background risk is and how it would change as you increase dose.
Q And, would you agree with me that that determination is based on the individual facts and circumstances of the

plaintiff's situation, at least in part?

MR. BERNICK: I'm sorry, just referring to Romanette 3, not the rest of the criteria later on for specific causation?

MR. FINCH: Yes, yes.

THE WITNESS: Could you repeat the question, I'm sorry.

Q In order to answer the question that Romanette 3 poses, you have to have data about the plaintiff's individual characteristics and that data is going to be dependent on the individual facts and circumstances of the plaintiff's situation, correct?

A Yes. The point I was making is that if you were going to take the dose response model and say where does a particular exposure group sit on that curve, you'd want to be reasonably sure that their characteristics match those of the population that you studied to get the curve. If they're different, I think I answered a question about alternative causes for specific individuals where you might have some other explanation. That's the point I was trying to make here, yes.

Q Then you write, about halfway down in paragraph -- the next paragraph, "Plaintiffs condition may be very rare in which the likelihood of demonstrating specific causation inquiry 4, increases." Do you agree if the condition which the plaintiff has is very rare, but there's general causation established

between the plaintiff's condition and exposure to a toxic chemical that it is the likelihood of being able to demonstrate specific causation increases?

MR. BERNICK: At this pont it really is unfair and incomplete. We've gone through three Romanettes, we now have an inquiry, Romanette 4, that Mr. Finch hasn't even put into the equation and the specifically referred to in that sentence. It's a very misleading question.

THE COURT: I think from -- well, this -- I mean, the doctor wrote the article, so he probably is able to ferret out whether or not the question is misleading and if it is, to ask that it be rephrased. It seems to me that the Romanette 4 does ask a specific question, and, let's see. Romanette 4, I do not believe is incorporated within your question. The objection is sustained.

Q Okay. Incorporating Romanette 4 within my question, do you agree, Dr. Rodricks, that if the plaintiff's condition is very rare, and he is exposed to a toxic chemical which is known to cause that condition, the likelihood of being able to demonstrate specific causation increases?

A Only in the sense that it would take -- for rare conditions it would generally take less exposure to double the background risk than for, say, a very common. Only in that sense.

Q On Page 60 and 61, you give a hypothetical example. I'm

looking at the second paragraph on Page 60, and you write, "Thus, for example, if a specific plaintiff's risk of developing his or her specific disease, in the absence of exposure to the suspect chemical is 1 in 1,000, examination of the dose response data from epidemiological data can reveal the magnitude of the dose necessary to increase risk by a factor of 1 in 1,000." Did you write that?

A Yes.

Q Okay. If the exposure experts can demonstrate that the plaintiff incurred exposures leading to a dose of at least that magnitude, what did you mean by that magnitude?

A One in 1,000.

Q Okay. Then it can be concluded that exposure to the suspect chemical increased plaintiff's disease risk to a level of at least 2 in 1,000, which is 1 in 500, right?

A Yes.

Q Thus, it may be concluded that since the plaintiff actually has the disease, then the risk has been realized and it is more likely that it was caused by the chemical than by whatever other factors caused the condition. Is that your view?

A That's what I had testify to earlier, yes.

Q Okay. Next paragraph you write, "This sketch of how the analysis of specific causation may proceed is meant to describe the type of analysis necessary, the scientific method to be

used and is not intended to describe a strict set of inflexible criteria (such as for example, a strict risk doubling standard). A degree of scientific judgment is necessarily involved especially since the data required to conduct a careful, quantitative evaluation of the plaintiff's exposures and epidemiological dose response relationships are almost never without uncertainty." Do you agree with that?

A Yes.

Q The next page, Page 62, you have a heading called Limits Improving Causation.

A Yes.

Q Do you have that section?

A I do.

Q The second paragraph, although the general process described here for undertaking an evaluation, you write "Although the general process described here for undertaking an evaluation of general and specific causation may have broad acceptance. It seems clear that nothing approaching the uniformity of scientific approaches that can be discerned in the regulatory context, exists in connection with the evaluation of individual exposures and responses. The relative weights given to different types of scientific information may vary greatly among experts and there appears to have been little substantive discussion of the problem of individual disease causation in the scientific as against the legal

literature." I take it you agree with that.
A I do, in the sense that that takes away nothing from my statement earlier that the general method of application here is perfectly appropriate, it does have consensus. There are going to scientific quarrels over the underlying data, what the best dose response might be. So there you will run into, perhaps, a lack of consensus, but the method to me is quite clear.
Q But there will be -- there could very well be lots of disputes about what dose the plaintiff was exposed to, or what dose is necessary is to have any increased risk of disease or whether you can determine specific causation in a particular case. Those are always going to be subject of expert disputes in an individual case.
A For working within --
MR. BERNICK: I'm sorry, excuse me. Those are three different questions.
THE COURT: Yes, they are.
Q Would you agree with me that there could be disputes in an individual case about the amount of exposure the plaintiffs cover, correct?
A The general -- when working with risk assessment methodology I described, is you look at the data and try to build models. Some scientists may come up with a slightly different model, or it could be a substantially different

model. There will be those kinds of scientific debates. I don't know about this particular case, but in some cases that happens. That has nothing to do with method, the method here is solid. And so, I was focusing here on the possibility that there could be a debate about some of the intricacies of this application.

MR. FINCH: May I approach the witness, Your Honor?

THE COURT: Yes.

Q Dr. Rodricks, do you have what's been marked Exhibit 589 in front of you?

A Yes.

Q This is an article that you wrote in 1997?

A Published in 98, yes.

Q Published in 1998?

A Right.

MR. BERNICK: Again, Your Honor, I can't help but point out, the title of the article is Toxicological Risk Assessments in the Courtroom.

MR. FINCH: Your Honor, there are statements in the article that go to his testimony here that have nothing to do with trying to provide a legal framework. There are statements about science.

THE COURT: Let's just -- Mr. Finch, we'll take objections to questions as they come.

MR. FINCH: Okay. Well, he objected to -- I thought

he was objecting to the article.

THE COURT: That was a statement, not an objection. Let's go.

Q Okay. On Page 24 of this article, you write, in Romanette 6, "If the observed toxic response is carcinogenicity, a different set of assumptions is adopted. High dose cancer studies at practicable size can detect excess lifetime cancer risks no lower than about 1 in 10, where as public health goals generally call for protective limits corresponding to very much lower risks. Extrapolation to the low dose, low risk range is thus require if ay statement about risk is to be made." Do you agree with that?

A Yes, that's what I said earlier in my testimony, basically.

Q And, in the United States a linear no threshold dose response model has been traditionally used, although alternative models, including threshold models have been advocated for some substances, based on data concerning their modes of biological action, correct?

A That is correct and it's truer 10 years later than it was then, yes. A lot of certain threshold hypothesis now (indiscernible), based on biological studies.

Q Could you turn to Page 28. The first full paragraph you write "With respect to epidemiology findings it is important to remember that population based epidemiology evidence can

establish only general causation, i.e., is the agent capable of causing the disease and not specific causation, i.e., did the agent cause disease in a given individual." I take it you agree with that.

A Yeah. If you find that something causes disease, you just can't assume that it always causes disease under every circumstance. That's why you need to proceed further.

Q Would you agree with the proposition that if there is no epidemiological evidence at all that shows that a given substance can double the risk of disease, then in making a -- let me back up. Here's my hypothetical. You've got exposure to substance A, which is a category 1 carcinogen listed by IARC.

A Okay.

Q Then you have exposure to some substance B, as to which there is no epidemiology at all that shows any doubling of the risk at any dose. As between the two, would it be sound science to totally discount the possibility that substance 2 could have contributed to the plaintiff's disease?

MR. BERNICK: Object to the form of the question insofar as it references IARC. That's number one. And, number two, I don't even understand the comparisons being drawn. Totally disregard this doesn't have any meaning to it. If we could have a precise question put that's not designed to back door the asbestos classification, maybe we could get an answer

to the question.

MR. FINCH: I didn't ask him about asbestos at all, Your Honor.

THE COURT: No, you didn't but you did ask about something that is obviously known to contain asbestos on a list, after having objected to this witness's qualification to answer the question. The objection is sustained. Restate the hypothetical.

Q Okay. Let me ask it this way. Assume that there are more than 10 studies that show for carcinogen A there's a doubling of the risk of cancer.

A I'm sorry, at a particular dose?

Q At a particular dose.

A Okay.

Q And, assume that for substance B there is no epidemiological evidence at all that shows that there is a doubling of the risk of the same disease that is caused by carcinogen A. Do you have that hypothetical in your mind?

A I guess what I don't understand is with substance B, is there any evidence showing it's a carcinogen at all below a doubling?

Q There is no evidence at all showing that it doubles the risk of disease.

A Does it increase the risk at all of disease. That's what I don't understand.

Q There's no evidence that it increases --
A So, it's not a carcinogen.
Q Correct.
A Okay.
Q There's no epidemiological evidence that suggests that it is. Some people have asserted that it is.
A Okay.
Q In that circumstance, would it be within sound science for someone who presents the disease, for their medical professional to totally discount the chances that the disease was caused by substance B? If they were exposed to substance A and there was more than 10 epidemiological studies that show that substance A can cause the disease and nothing that shows that substance B can cause the disease.

MR. BERNICK: Again, I --

THE COURT: It's all right, Mr. Bernick, the witness can answer this question.

THE WITNESS: Well, in spite of --

THE COURT: If you can understand it, you can answer it.

MR. BERNICK: Okay.

THE WITNESS: I'd have to kind of restate it to be sure -- I think you're saying we have a well established carcinogen, we know the risk doubling dose, we know the clear evidence of carcinogenicity, and there's some other substance

where we have no evidence of carcinogenicity, now, you're saying if somebody -- well, I don't see how B is relevant to any question. Maybe I just don't understand it, I'm sorry.

Q No, the question is, if someone in making a specific causation determination decides that there is no way that substance B could have possibly caused or contributed to this person's cancer, that would be within the realm of sound risk assessment science.

A Given the facts you stated, yeah, you wouldn't say that B had any risk of cancer, right. Any known risk of cancer.

Q On Page 29, under number 3, the bottom of the first paragraph you write: "It is nevertheless obvious that reaching a scientific consensus on the methods to evaluate chemical dose is not to be expected, especially when the objective is to estimate doses to specific individuals and not to generic populations. Indeed, some experts offer the view that the magnitude and duration of dose do not matter, especially for carcinogens, the mere fact of exposure is sufficient to conclude the presence of increased risk or in some cases actual causation." Did you write that?

A I don't see where you're looking. I mean, if you're quoting me, I wrote it. Now, the question is, I hope I discounted that idea.

Q You hope you what?

A Discounted that idea as incorrect. Could you tell me

where you're reading here?

Q Yes, at the -- it's highlighted on the ELMO.

MR. BERNICK: Give him the page, Page 29.

Q Page 29, paragraph 3.

A I'm just having -- I apologize. Oh, okay, I'm sorry. It's the first paragraph under --

Q Yes.

A Okay.

THE COURT: This is talking specifically about levels of lead in the blood?

MR. BERNICK: Well, even more than that. If you read up further in the paragraph, it's about -- basically it's a statement about a problem that experts who are toxicologists have trying to be experts in dose reconstruction. I don't --

Q You're a toxicologist, correct?

A Yes.

Q All right. Would you agree that there is not a scientific consensus on the methods to evaluate chemical dose?

MR. BERNICK: Your Honor, I would specifically and vehemently object to that question. It is totally misleading because he's taking it out of context in an article that's all about experts who are called in litigation. This is an improper use, it's not proper impeachment.

THE COURT: It is an improper use of this article, but I think if you'd heard the witness' answer, you probably

wouldn't be objecting of vehemently. Nonetheless, the objection is sustained.

Q All right. Let's turn to Page 30. Do you agree that most toxicologists would not adopt the view that doses already documented as toxics are the only doses likely to cause toxicity?

A Where are you at, I'm sorry?

Q I'm just asking him in general, does he agree with the proposition that most toxicologists would not adopt the view that doses already documented as toxic are the only doses likely to cause toxicity?

A I'd be careful with the word cause here. I wrote that, but I was referring to what I was asked about earlier, that you measure -- you fail to find effects, you believe that the effects simply disappear at the point where you failed to find them, and I think most would say, well, there's going to be some risk of toxicity as you go below those doses, we don't know what the shape of the dose response curve is, we don't know how quickly they go to zero, but qualitatively we might say there's some risk but I think it's going to be a very small risk and I think I tried to say that earlier. That's the point I was trying to make here.

MR. FINCH: May I consult with my colleagues for a second?

THE COURT: Yes, sir.

(Pause)

MR. FINCH: Your Honor, I would just offer at this time the two articles, 589, and what's the other one?

UNIDENTIFIED MALE SPEAKER: 588.

THE COURT: 588.

MR. FINCH: 588.

THE COURT: They're admitted?

MR. BERNICK: No, well --

THE COURT: They're the witness's articles.

MR. BERNICK: They are the witness's articles but the fact that the witness authored the articles doesn't make them any more admissible as underlying materials for expert testimony. Underlying materials for expert testimony --

MR. FINCH: I'm not offering --

MR. BERNICK: Excuse me. Are inadmissible. The fact that they're used on cross-examination and that he wrote them, doesn't make them admissible. I mean, they're only used for impeachment purposes and then you quote them, use them for impeachment purposes, the rest of the article doesn't come. in.

THE COURT: Well, that is a fair statement and, frankly, there wasn't much impeachment material, based on what this witness testified to. He was basically affirming what he had testified to on examination. So, I'm not sure what the purpose is. I would think, however, Mr, Bernick, based on the fact that this Court cannot take 100 percent accurate notes,

and at some point I know I'll get a transcript, but I don't know when, I would at least like to have the pages referred to by the witness.

MR. BERNICK: I would -- Your Honor, and the only reason I stand on what I think is an appropriate technical objection is, I'm just very sensitive about establishing precedence in the case that will then lead to people offering all kinds of stuff in, just because it was used on cross-examination.

THE COURT: Well, and I agree.

MR. BERNICK: I'm happy to have Your Honor have the whole thing.

THE COURT: I agree with the principle, if it's not substantive evidence, but to the extent that somebody is going to refer to it as a portion of cross-examination, I would like a reference point to have it available, but I agree.

MR. FINCH: With that amendment, then I would offer for purposes of impeachment, the sections of the article that I used with him on cross-examination, not the entire article.

THE COURT: Well --

MR. BERNICK: You don't offer -- again, they are not offered separately. They are used for impeachment, or they're not and whatever you read out of them is the impeachment but, again, I have no quarrel with the Court having, indeed, the full document so that the Court doesn't have any confusion

about what it is that was said on the record.

MR. FINCH: That's my only concern, Your Honor. I just want the Court to be able to refer to the actual article that I used, even if it doesn't substantively come into evidence.

THE COURT: That's what I would like to be able to use the articles for, not as substantive evidence, but simply to make sure that I do have a complete record because it's going to be very difficult as we get through 18 days of this, to keep track of what witnesses are referring to what. So, they will not be admitted into substantive evidence. I am going to maintain copies in the file so that I do have a record of the specific portions that were referred to by any one in this case and the witness. All right.

MR. FINCH: I'll pass the witness, Your Honor.

THE COURT: All right.

MR. RASMUSSEN: Good evening, Your Honor.

THE COURT: Good evening.

MR. RASMUSSEN: I'm Garret Rasmussen for the Future Claimants. My first time in the court, so I'm happy to meet you. And Dr. Rodricks, my first time to meet you as well. I think we'll probably have a lot to agree upon and my cross-examination will not be a long one.

CROSS-EXAMINATION

BY MR. RASMUSSEN:

Q I want to start out by asking you some questions about risk assessment and the relevance of risk assessment to the evaluation of disease in individuals. So, risk assessment, first of all, focuses on likelihood of adverse affects occurring in exposed populations, correct?

A It will vary, it can do that, yes, for sure.

Q Right. And a populations risk of a disease is not the same thing as the risk to each and every individual in that population, is it?

A It's not identical, no.

Q Correct. A populations risk is only the same as every individuals risk is all the individuals have identical exposures and identical sensitivities to the particular toxin, isn't that right?

A Well, not exactly that. We would say that if we're talking about a group of individuals who experience exposures similar to those where we have observation of excess risk, and there's nothing that would distinguish them otherwise from the studied groups, then what you find for the population ought to apply to those individuals, but it applies in a probabilistic way. I think I described that with the drug example earlier. That was my testimony and that's what I believe.

Q Would you agree, Dr. Rodricks, that regulatory risk based standards are not useful to evaluate disease causation in particular individuals?

A They are not designed to do that, they should not be used for that purpose.

MR. BERNICK: Regulatory -- counsel, the question is regulatory risk levels?

MR. RASMUSSEN: Yes, yes. Yes, it was.

Q And, it's true isn't it, that the assumptions and models used in regulatory risk assessment are not known to apply to any actual individuals, correct?

A They are not designed to work in that way. They try to focus on hypothetical, most sensitive individuals, whoever they are, we don't know who they are, most highly exposed. So, they have a number of assumptions that would be very -- it would be just wrong to apply to individuals, when you're dealing with those assumptions.

Q Okay. So to assess the disease causation in an individual, you would have to look at that specific individuals exposure, isn't that right?

A Yes. Among other things, but yes.

Q Yes, it's not enough just to look at a group cumulative exposure, is it?

A Well, you could do it on a group basis as well. If you have -- the important thing is the general causation and the dose response relationship, but now we could look at some group that has some range of exposures and that you could --

Q But you couldn't do it --

MR. BERNICK: Excuse me, let him finish.

MR. RASMUSSEN: Yes. Keep going.

THE WITNESS: What I'm saying is, you could do it. Often cases arise where there's an individual or cases maybe small groups of individuals, or large groups who have similar exposures. So, you could do it in all of those -- I'm sorry. Go ahead.

MR. RASMUSSEN: Finish, please finish.

THE WITNESS: Well, you could to it in all of those circumstances.

Q Are you familiar with the concept of a heterogenous group?

A Well, it has a couple of meanings. Those variability in the population, yes.

Q So, if it is a heterogenous group with variability in the population, then you can't use the group cumulative risk exposure to make a prediction about an individual, could you?

MR. BERNICK: Objection to the form of the question. It's very ambiguous and it's got several elements to it. If you understand, go ahead and answer, but I --

MR. RASMUSSEN: I think it's actually a very precise questions and asked very precisely and I think the witness understands it.

THE COURT: Well, would you repeat it for me because I only got half of it. A heterogenous group with variability in a population, that's how it started comma, cannot use and

that's where it lost it.
Q Okay. Cannot use a cumulative -- cannot use a risk exposure calculated for a group, as opposed to for an individual. For a population as opposed to an individual.
MR. BERNICK: That same -- it's even worse now.
MR. RASMUSSEN: Well, we'll get back to that -- we'll start with that general question and we'll get more precise as we go on.
THE COURT: If you understand this question, Doctor, you can answer it.
THE WITNESS: I thought I did but now I'm not sure, so maybe it needs to be repeated.
Q Okay. Let me come about it a different way.
A Okay.
Q I mean, I'll just start asking about cumulative exposures because I think I really want to get to the cumulative exposure point.
A Okay.
Q The point that you did understand. So, you would agree that there's a positive relationship between cumulative asbestos -- well, I don't want to use asbestos. That's a word I can't use. You'd agree that there's a positive relationship between cumulative exposure to a toxant and a risk of getting a disease because of that exposure?
MR. BERNICK: I'm sorry.

THE WITNESS: That's the first premise?

MR. RASMUSSEN: Yes, that's the first premise.

MR. BERNICK: He's being asked to assume that as a hypothetical?

MR. RASMUSSEN: No, I'm asking him if he agrees with that.

MR. BERNICK: I don't know what he's agreeing with.

MR. RASMUSSEN: Well, let's ask him.

MR. BERNICK: What --

THE COURT: The question is, does he agree that there is a positive relationship between the cumulative exposure to a toxant and the risk of getting a disease from that exposure, but I'm not sure what the cumulative exposure means. I'm sorry.

Q Okay. The exposure -- the dosage is really what it means.

Q Yes, yes.

THE COURT: But you were talking about heterogenous populations. You've backed off that and now you're talking about individuals with more than one --

MR. RASMUSSEN: No, I'm doing it step by step. I'm going to get back to the heterogenous population, but I want to first start out with the concept of what dosage means.

Q Dosage -- and --

THE COURT: So, now, you're now talking about an individual with more than one exposure. That's what you're

talking about dose, cumulative exposures?

MR. RASMUSSEN: No, no, I don't want to talk about -- we're getting ahead of ourselves. I'm going to go back and lay the foundation.

THE COURT: All right.

Q Each individual, individuals in a group can have different exposures to a toxant, correct?

A There would be variability, yes. Well, a group, when you say a group, let's say who are all doing one kind of job exposed to a chemical, there would be some variability on any given day, right.

Q Right. And also there could be some variability cumulatively over the course of a year if they were doing different jobs, isn't that right?

A If their jobs were -- quite different jobs with the same chemical, let's say?

Q Yes.

A Sure. Yes. So, we'd want to classify, if there were different jobs, you'd probably want to look at them separately.

Q And sometimes even people doing the same job could have different cumulative exposures, not just a daily exposure, but cumulative, over the course of a year if, indeed, they do the same job in a different way.

MR. BERNICK: These are all hypothetical questions, not grounded -- you're asking him to assume that?

THE COURT: They are hypotheticals, yes.

MR. RASMUSSEN: Correct.

THE WITNESS: Yes, I don't know the answer to that. I mean, that really gets to be, say, an industrial hygiene or chemists question.

Q I'm not asking you whether that could happen, but if it did happen --

A You're assuming that it does happen.

Q Yes, yes.

A Right.

Q And, if it did happen, then the exposure of an individual could differ from the exposure -- well, then the risk to the individual could differ from the average risk to the group, isn't that right?

A Given your hypothetical --

MR. BERNICK: I'm sorry. The question is whether an individual may be -- an individual's exposure may be different from a calculated average, is that the question?

MR. RASMUSSEN: Yes, yes.

MR. BERNICK: You're asking whether it can possibly happen?

MR. RASMUSSEN: Whether it can happen in a group of people, each doing the same job, because they're doing the same job in a different way, over the course of a year, not just in one day.

MR. BERNICK: I think this really, again, falls outside of his scope of expertise, but if he can answer it, it's very vague.

THE WITNESS: I don't really -- this really is beyond my real experience. I mean, I know there's variability from day to day, I know that when I look at exposures as a risk assessor, it's usually the long term exposure, either for a certain group because that's usually the best measure of the average exposure over a long period of time, if it's that long term exposure that matters, and in most risk assessments that's what matters.

There may be variability around that day to day for individual to individual, but if you're looking at the group, it's that long term average that is the best, most probable. We're talking about what's most likely to be correct. It's not actually every single individual. That's my point about going from groups to individuals, we're not talking about absolute prediction, we're talking about what's more likely than not, or more probable than not. I'm sorry, I kind of lectured, I apologize.

Q No, no, that's fine. But the individuals in that group, an individual in that group could have a cumulative exposure, in other words a long term exposure over the course of a year that differed from the average exposure of that group, over the course of the year.

MR. BERNICK: Your Honor, I just --

THE WITNESS: You can't deny that, sure.

THE COURT: If that weren't possible, we couldn't get an average.

MR. RASMUSSEN: Beg pardon?

THE COURT: If that weren't possible, we couldn't get an average.

MR. RASMUSSEN: Right. That's my point.

THE COURT: Okay, so let's move on to something.

MR. RASMUSSEN: So, some people would be higher than the average and some would be below.

THE COURT: So, let's move on.

MR. RASMUSSEN: Okay.

MR. BERNICK: (Indiscernible) what an average means.

Q Relative risk. In order to tell whether anyone in an exposed group has sufficient cumulative exposure to a toxant to achieve a risk doubling exposure, you would want to look at the distribution of cumulative exposures in the group, wouldn't you?

A I'm sorry, could I -- I don't want to have the question read back. Could you just restate it, I'm sorry.

Q Yes. In order to -- with respect to relative risk, in order to tell whether anyone in the exposed group, exposed group to a toxant, had sufficient cumulative exposure to the toxant to achieve a risk doubling exposure, you would want to

look at the distribution of cumulative exposures in that group, correct, so you'd know whether some people are above the average and some might be below.

MR. BERNICK: Objection. I think that is -- this is only pursuing the same question which is the degree of variability of exposures in a group. And, again, I think it's outside the scope of his expertise. I know it's outside the scope of my examination. He's trying to use this individual to create a predicate for a declaration that Dr. Stallard introduced in this case, too late in the day, and this actually violates yet again, yet again, the agreement that was reached, that the testimony by declaration of Dr. Stallard would be used solely for Daubert purposes and would never make its way into this trial. And now it's not only being done back door through cross-examination of a witness, it's not even in the area.

THE COURT: All right. This witness has a good understanding of his own expertise. If he thinks this is outside the area of his expertise, he has been very quick to state that. It seems to me that if this is outside the area of his expertise, he will so state and otherwise, if he understands the question, he may answer it. Doctor, you may answer this question if you understand it and feel that it is within your expertise.

THE WITNESS: Well, I'd like to hear it one more time, though, because that discussion, I lost the question.

Q Sure, okay. In order to tell whether anyone in an exposed group had sufficient -- any one person in the exposed group, had sufficient exposure to a toxant to achieve the risk doubling exposure, you would want to look at the distribution of cumulative exposures in that group, wouldn't you?

A You're talking about a single person --

Q Yes, I am.

A I don't see how a distribution helps you deal with a single person.

Q If you want to try to figure out the causation for an individual, the range of where an individual might lie, if you want to figure out the extent to which an individual might have a cumulative exposure over the course of say a year, and you had an average for the course of the year, you'd want to know the variant, the variation in order to tell how high the individual might be or how low the individual might be relative to the average.

MR. BERNICK: Determining that variation for what purpose?

THE COURT: That was not a question, that was a statement. There is no question before this witness. He has said he doesn't think that's a significant factor, that was his answer. Could we please get a question before the witness?

MR. RASMUSSEN: Okay.

THE COURT: The statements are not evidence and

they're stricken.

MR. RASMUSSEN: Okay. Let me ask the question again, or ask a question, I'm sorry, Your Honor.

Q In order to tell whether anyone in the exposed group had sufficient cumulative exposure to a toxant to achieve a risk doubling exposure --

THE COURT: You've asked that question. He's answered it.

MR. RASMUSSEN: Okay. Did we get --

Q Okay. If we've got the answer then I'll move on.

A Well, I'll answer again if it helps. I mean, I think the answer is no, that if you have a distribution the average -- you don't know where any individual is on the distribution, they could be on the high end, or the low end, the average is the best measure over a long period of time. If you had to say what is the most likely exposure for an individual in that group, you'd say it's the average. I'm quite sure that's right.

Q Well, if you knew the average cumulative exposure of the group, you couldn't tell from that whether any particular individual within a group would have sufficient cumulative exposure to the toxant to contract a disease because of that exposure, could you?

MR. BERNICK: Objection. Wouldn't -- well answer it if you can.

THE WITNESS: I'm working on what's probable. The average exposure for a group is te most probable exposure and calculating the risk based on that exposure makes perfect sense for the group and you would then say, probabilistically that is the best estimate for any individual in that group. The only thing that would exclude that analysis was some other factor about some individual which makes -- which says you have evidence that that individual was somehow very different from the rest.

Q But there may be individuals in that group that will have a cumulative exposure that is greater than the average, that's my point.

MR. BERNICK: Your Honor, we've been through that like three times.

MR. RASMUSSEN: Okay. If that point is established then I'll move on. Do you agree with -- do we have agreement on that?

THE WITNESS: I suppose that could not be discounted, but you're asking to do an assessment of the risk for the group and what is most likely by way of exposure. Unless you have some other evidence, what is most likely is the average.

Q But I'm asking for the individual. I'm saying yes, that's the average for the group, that's the most likely for an individual in the group, but I'm asking you whether at the same time there will be individuals in that group who will actually

have a cumulative exposure that is above the average.
A Right. And I'm saying it doesn't matter.
MR. BERNICK: Objection, that calls -- I'm sorry. That calls at this point, whether -- complete speculation.
THE COURT: Counsel, Doctor, in order to get the average, as a mathematical proposition what do you have to do?
THE WITNESS: Well, you have a job, you have a chemical, you send in an industrial hygienist, they measure daily or weekly, they do this over a long period of time for people working that job, they can measure the direct breathing zone or just the general area and they make an average. And you'd say for that job, unless you had some clear evidence that something was very different for some employee, you'd say for that job the average exposure is what determines the employee's risk, on a probabilistic, you can never be absolutely sure, but on a probabilistic, what's most likely to be true, the average is most likely to be true.
THE COURT: And, sir, if a range of exposures for any particular individual within that group, hypothetically, ranged from 1 exposure in a given period to 100 exposures in that same period, and you took an average and the average exposure was 50, is it a fairs statement that there would be some people who had less than 50 exposures, and some people who had more than 50 exposures?
THE WITNESS: There may be on any given measurement

day.

THE COURT: Yes.

THE WITNESS: But on the long term, unless there were some other circumstance that all ought to sort of balance out and the --

THE COURT: To the average.

THE WITNESS: -- and the average is the best most probable estimate of their exposure. Yes.

THE COURT: Thank you.

Q But, it wouldn't balance out to the average over the long term if the individual in the group were doing different jobs within that job category, would they?

A Well, that's a different matter, yes.

Q Well, it's true, isn't it? If they --

MR. BERNICK: Objection, objection. Your Honor, at this point, I really think he's badgering a witness who has gone as far as he can in answering the questions.

THE COURT: That question has been asked and answered.

Q Suppose that the risk doubling dose for toxant is 2, just as you have in your graph this afternoon, you can't tell whether any particular individual within a group would have a sufficient cumulative exposure to the toxant to contract a disease simply knowing that the cumulative average was sufficient to get you to a 1.9 relative risk, could you?

MR. BERNICK: Objection. This exact same question was asked 15 minutes ago, and led down this whole path and I really object, Your Honor, because this, again, is an effort to use an analysis, now on cross-examination, that was specifically agreed to be usable solely for Daubert purposes --

THE COURT: Well, I don't know that. It seems to me that this whole line of questioning, this witness has been very clear, we're going over ground that this witness is not changing his opinion about. Can we get to something new?

MR. RASMUSSEN: I really would like the answer to that question because I think, Your Honor, we'll find out later in this trial that that question is a key question and that other --

THE COURT: Then if that is the key, you may establish it in your case. This witness has told you that he is not measuring individual populations. Now, you keep asking him about individual populations.

MR. RASMUSSEN: But I'm asking him about -- he's an expert on risk assessment --

THE COURT: Yes, he is.

MR. RASMUSSEN: -- and his testimony is how you use risk assessment and, indeed, he has a title in his report called Relevance of Risk Assessment to a Valuation of Disease Causation in Individuals.

THE COURT: Yes, he does.

MR. RASMUSSEN: And I'm asking him about causation in individuals based on risk assessment. And I'm asking him about the impropriety of using group, or at least the distinction, maybe not the impropriety, the distinction between using a group relative risk to apply it to an individual and I think --

THE COURT: And he has been very consistent in answering those questions. You may ask one more question along these lines. Ask your question, again. The doctor may answer it, he is not changing his testimony about this.

MR. RASMUSSEN: Okay. One last question on this line, it's a very important question. I'd like to have an answer, I think it's a fair question.

Q Suppose the risk doubling dose for a toxin were 2 units, which is what you testified about earlier this afternoon, and the average cumulative lifetime exposure of a group were such that the relative risk for the group was 1.9, which is below the doubling dose standard, even though that's below the doubling dose, you couldn't conclude that nobody in that group had an exposure that could exceed the doubling dose and, therefore, have causation, could you?

A I can only -- it's the same answer I really gave to the last exposure question. If you're asking what is most likely, the average exposure for the group is what's most likely for any individual, most likely, it's not perfect, but most likely, and you would say for a 1.9 increase that you do not meet the

more likely than not standard.
Q My question wasn't whether it's more likely for the group, it was whether knowing that the relative risk for the group is 1.9, you could not conclude from that knowledge that nobody in the group exceeded the double dose of 2 units.
A Well, I could not do that if you're seeking perfect knowledge, right. In other words, if it's only a matter of absolute knowledge, then I can't. I can talk about what's most likely.
Q But isn't it common sense as the Judge said, you have an average and some people are going to be above the average and some are going to be below the average?
A Well, I don't agree.
MR. BERNICK: There's been almost no common sense in this line of examination. Your Honor, we've been over this now for 25 minutes.
THE COURT: I believe the relevant test is, what is the accepted scientific standard in the community within which this witness is permitted to offer an expert opinion. So, why don't we get back to the test and ask your question within the framework within which he is permitted to offer an expert opinion. If it's to a reasonable degree of scientific certainty that you're trying to get him to answer a question, then, perhaps that is the question that needs to be asked.
Q Okay. I'll do that. Suppose the risk doubling dose for a

toxin were 2, and suppose the average cumulative lifetime exposure of a group was such that the relative risk was 1.9, could you conclude with a degree of scientific certainty and reasonableness that a person, that nobody, that no person in that group exceeded the relative risk doubling dose of 2?

A You're assuming the 1.9 is well validated as the average and are you asking me to conclude with absolute certainty that there's no --

Q No. Reasonable scientific certainty was my question.

A All I can answer is the most scientifically responsible and certain answer is that the average is what affects the risk for the group. And I couldn't go -- and that the most likely exposure and risk for every individual, the most likely is reflected by the average.

Q So, therefore --

A I don't know what else to say.

Q So, therefore, am I correct in concluding that you cannot say that nobody in that group exceeded the relative -- the risk doubling dose of 2.

MR. BERNICK: Your Honor, this is really --

THE COURT: It's sustained. He has now told you everything he is going to tell you.

MR. RASMUSSEN: With all due respect, Your Honor, I think he answered a different question. If you read back the question and read back the answer, you'll see he didn't answer

the question, he answered a different question and talked about the group.

MR. BERNICK: With all due respect, this shows disrespect for this witness and this process and I think we're creating a very dangerous precedent in this case.

THE COURT: Counsel, he has answered to the best of his ability. You can keep asking this question until the cows come home, you're not going to get a different answer. The objection is sustained. You have tried at least 15 ways to get this answer -- this witness to give you a different answer, he's not giving you a different answer, I'm sorry. You've got to live with the answer he's given you.

MR. RASMUSSEN: Okay. That actually brings on the next question, it makes the next question even more appropriate.

Q Would you agree, sir, that risk assessors are, perhaps, most delinquent in their failures adequately to convey the uncertainties that accompany their evaluations?

A I think I probably wrote that.

Q So, you'd agree with it?

A Yeah.

Q And, you would agree that risk assessors should report the range of uncertainty when a risk assessor reports her conclusions wouldn't you?

A I'm sorry, I didn't hear -- could you please repeat the